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BOSTON

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# INCORRECTNESS OF THE DIAGNOSIS OF DEATH FROM INFLUENZA

## PRESENCE OF BRONCHOPNEUMONIA IN PRACTICALLY ALL PERSONS SEVERELY ILL WITH INFLUENZA *

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Boards of health, as indicated by newspaper reports, very generally are subdividing their mortality reports from epidemic influenza into deaths from influenza and deaths from pneumonia or bronchopneumonia. This is no doubt based on the death certificate return handed in by the physician in charge of the patient. Is it a correct return? In my opinion it is not. The correct return should be bronchopneumonia and (epidemic) influenza. Whether the bronchopneumonia is part of the influenza or is a complication of that disease; whether the disease is due primarily to the influenza bacillus; whether the pulmonary consolidation is caused primarily by the *Bacillus influenzae* or is the result of concomitant bacteria are questions that cannot be answered on the basis of our present information. However, my own observations on patients suffering from epidemic influenza do justify a vigorous protest against a statistical subdivision of death reports into those dying from influenza without pneumonia and those dying from bronchopneumonia or pneumonia and influenza.

My reasons for believing that in practically all fatal cases of epidemic influenza there is a pneumonic process in the lungs before death are as follows:

1. In 126 consecutive fatal cases of epidemic influenza observed by me and my assistants at the Peter

* From the Medical Clinic of the Peter Bent Brigham Hospital.

Bent Brigham Hospital, not a single patient failed to show physical signs justifying a clinical antemortem diagnosis of bronchopneumonia.

2. In twenty-three consecutive necropsies at the Peter Bent Brigham Hospital in fatal cases of this group, no single case failed to show pathologic changes in the lung justifying the diagnosis on the part of the pathologist of bronchopneumonia.

3. In patients submitted to necropsy, pulmonary changes are as a rule more extensive than physical signs during life had indicated.

4. Clinical study of nonfatal cases of epidemic influenza justify the belief that with very few exceptions, patients with fairly severe to severe illness have bronchopneumonia.

In making the foregoing statements I do not deny that influenza patients may die from an overwhelming toxemia without pulmonary involvement or from influenzal meningitis or encephalitis or from some other manifestations of the disease. Such fatal cases did not occur at the Peter Bent Brigham Hospital, and in conversation with my colleagues who have had postmortem experience in this disease I have found that their views have seemed to coincide with those expressed here by me. In our admissions to the hospital of patients sent in with the diagnosis of influenza, I have seen cases of meningitis; one case was due to the meningococcus and apparently did not have any complicating influenza; two patients were tuberculous and may have had influenza, and two cases were due to the pneumococcus in influenza patients with pneumonia. Several patients with influenza and having signs and symptoms suggestive of meningitis showed spinal fluids with normal cell counts and negative cultures. No spinal fluids in our cases showed *B. influenzae.*

The statement made under No. 4 is based on these facts. In 195 unselected cases[1] of epidemic influenza studied in the wards of the Peter Bent Brigham Hospital, 132 showed during their period of observation in the hospital sufficient physical signs to justify the clinical diagnosis of bronchopneumonia. The 132 include practically all severely ill patients and many

1. This represents only those patients discharged and having their histories completed and filed in the record room to date. The total of our cases, about 500 in number, will show somewhat the same rates.

who were only mildly ill. The only patients in whom the temperature was as high as 101 for more than three days in this series in which no diagnosis of bronchopneumonia was made were fourteen in number. Two of these had complicating otitis media, and one was an asthmatic with chronic bronchitis antedating influenza; eight were patients admitted on the day of their first symptoms, two on the third day of their disease, and one on the fourth day. Of these last eleven patients, none were more than mildly ill, and they appeared to be free of any complications.

The physical signs on which was based the diagnosis of bronchopneumonia were areas of bronchial breathing or consonating râles, usually both, frequently bronchophony and often dulness on percussion. The occurrence of any one of these signs in a localized area appears to justify the diagnosis of bronchopneumonia. In a number of our patients, roentgen-ray examinations were made and invariably showed evidence of consolidation where these physical signs had been observed. Moreover, the roentgen plate as a rule showed more extensive areas of consolidation than clinical signs indicated; and not infrequently it showed consolidation before we had felt justified in diagnosing it on the basis of physical signs. The peculiar snapping consonating quality of the râle in epidemic influenza seems of great significance as indicating consolidation. All who have been listening to the chest in influenza patients will, I am sure, recognize what I mean, though my word picture of the quality of the sound is very inadequate.

The early foci of consolidation in influenza are found almost invariably in the region of the angle of the scapula and the interscapular regions. Here the roentgen ray shows that consolidation begins, i. e., toward the inner lower border of the lung posteriorly rather than anteriorly. From such an early beginning, rapid spread often takes place, leading to very extensive diffuse consolidation and not infrequently giving the clinical picture of a lobar pneumonia, though I believe true lobar pneumonia is really rare in association with influenza.

If the busy clinician—and never were our medical men so overwhelmed with work as in this epidemic—will percuss rapidly the lower lobes in the back and

listen in the region of the angle of the scapulae and next in the interscapular regions he will find quickly in most severe cases evidence justifying him in diagnosing bronchopneumonia. Of course, in many cases further examination will yield evidence of much more extensive pneumonic involvement.

I should not be justified in making the extreme statement that all patients with epidemic influenza have bronchopneumonia. Many milder cases certainly give no physical signs of consolidation, and the roentgen ray shows no shadow sufficient to justify the diagnosis of a focus of consolidation. I believe, however, that we are justified in regarding epidemic influenza as a disease involving the respiratory tract and except in the milder cases causing a clinically demonstrable bronchitis and bronchopneumonia in the larger proportion of cases. That as the severity and duration of the disease increases the percentage of patients with bronchopneumonia increases, and that in fatal cases almost without exception bronchopneumonia is present, are conclusions which seem fully justified by the data here presented. A corollary to this is that, in my opinion, it is quite incorrect to consider fatalities in this epidemic as due to influenza uncomplicated by bronchopneumonia except in such exceptional cases as to form a negligible factor in statistical reports of death returns.